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Axon Guidance Defects in NFATc2/3/4 Mutant Embryos

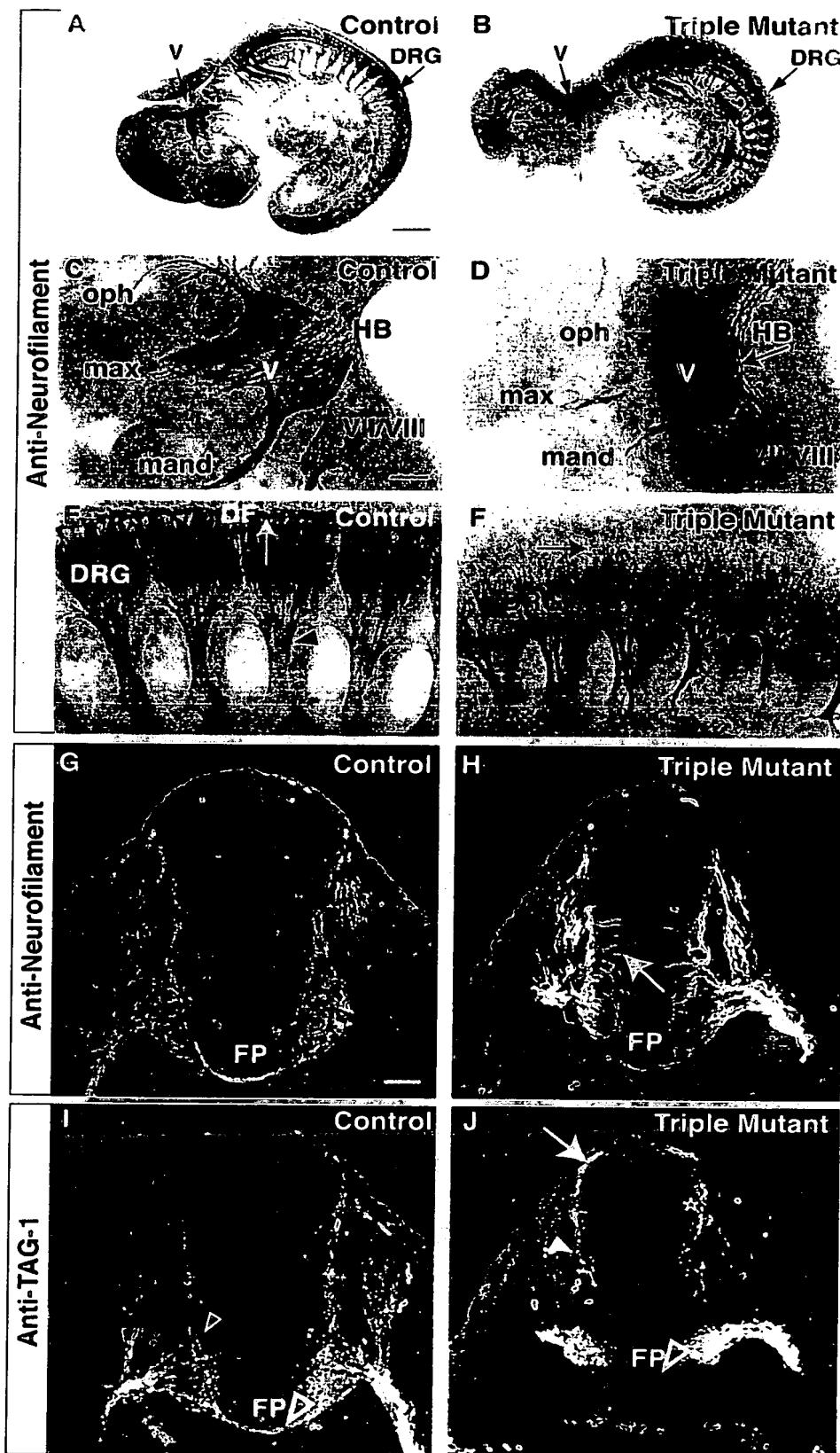
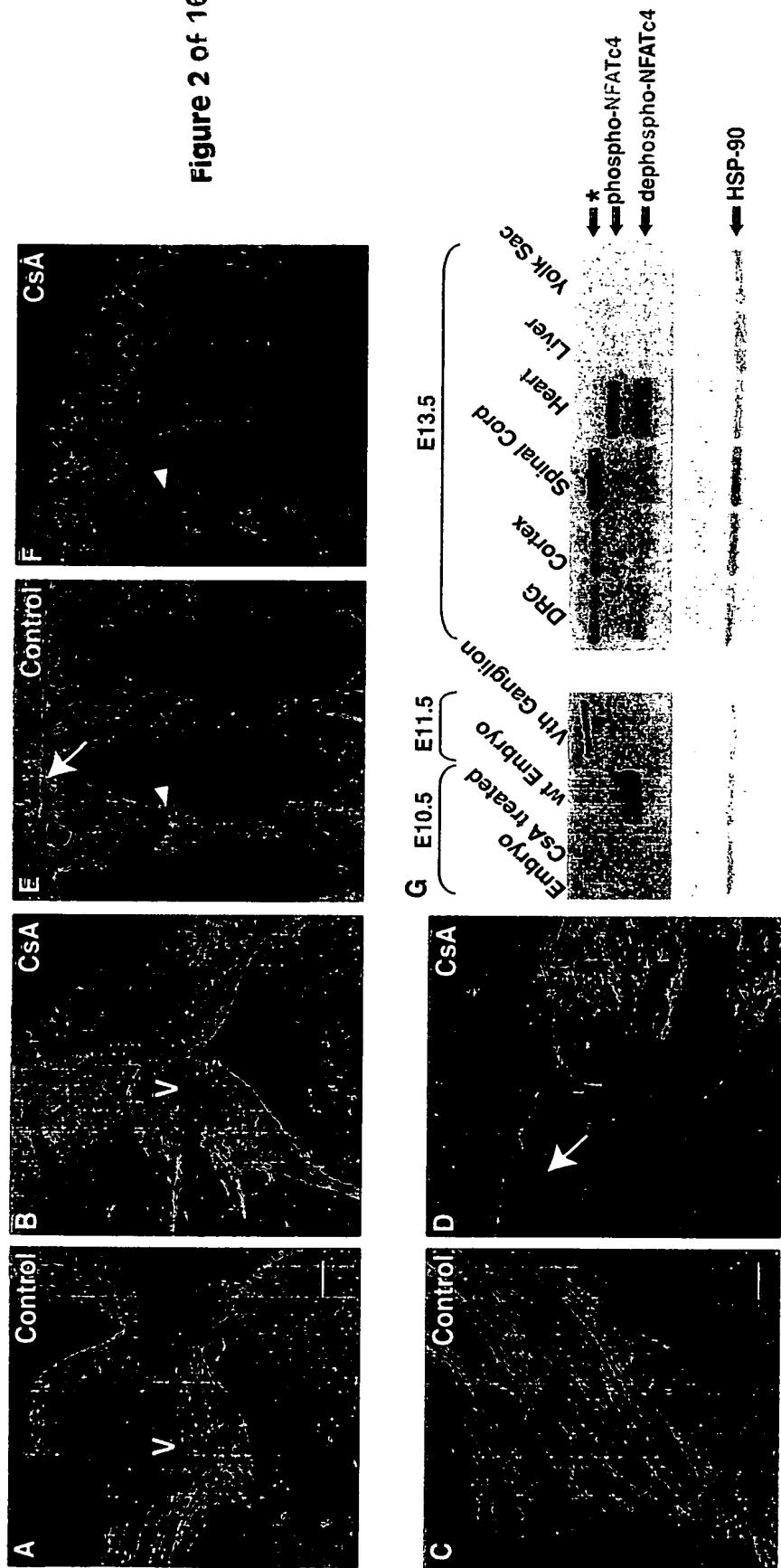


Figure 1 of 16

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# Pharmacological Calcineurin Inhibition during Embryonic Development Produces Defects Similar to those in NFATc2/c3/c4 Mutant Embryos.

Figure 2 of 16



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Cell Autonomous Defect of Sensory Axon Growth

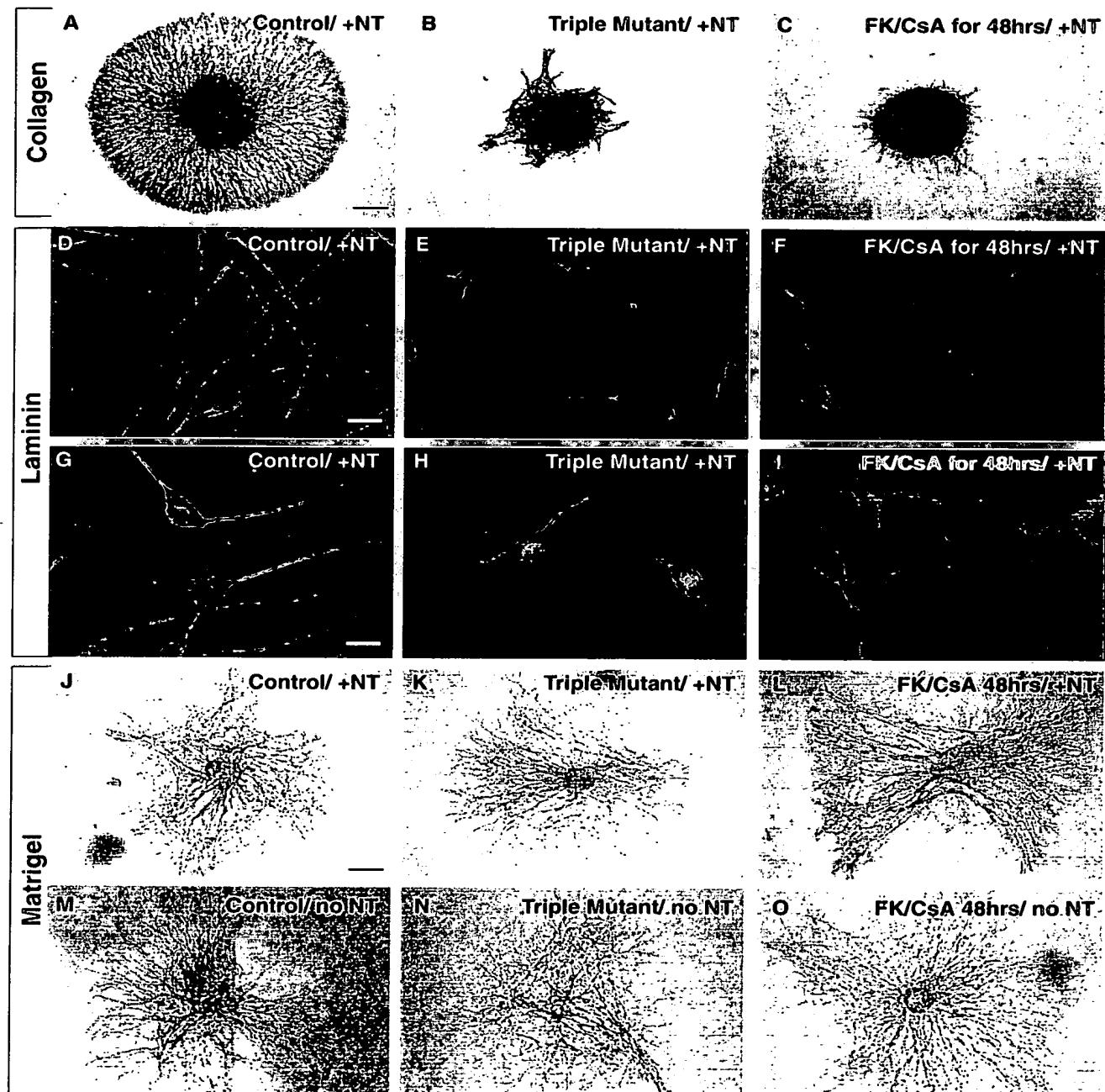


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Neither Calcineurin nor NFATc2/c3/c4 are Required for  
Sensory Neuron Survival *in vivo* or *in vitro*

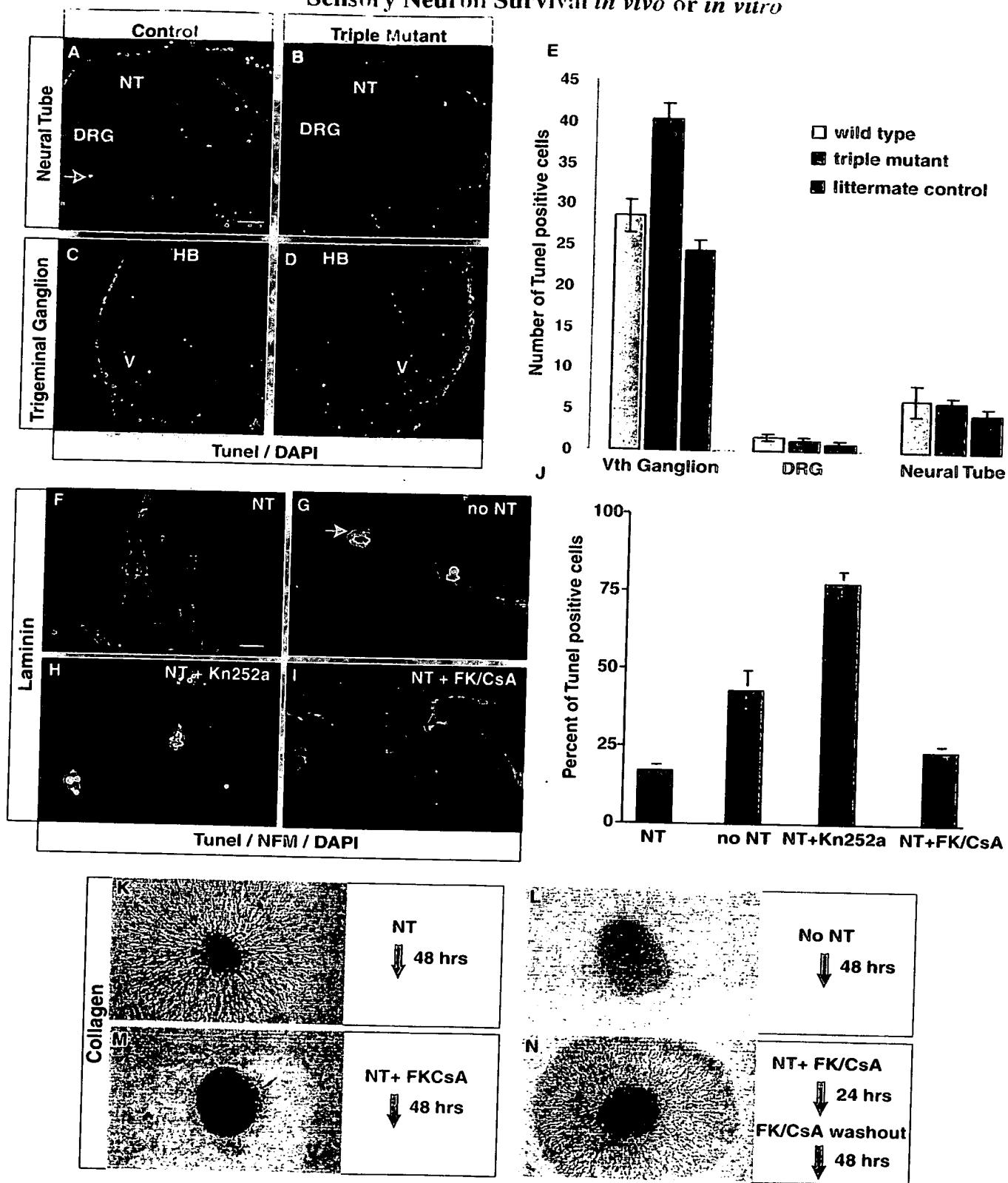
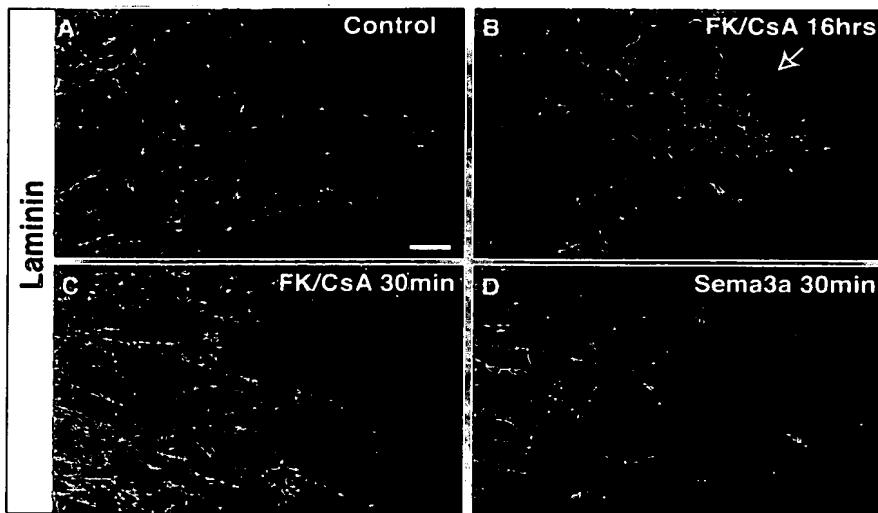


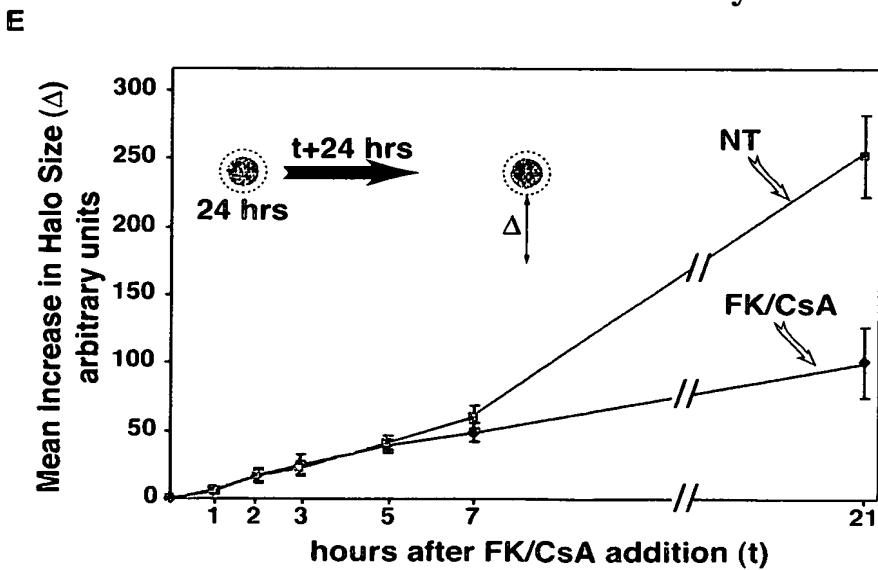
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**Calcineurin Inhibition does not Produce  
Growth Cone Collapse**



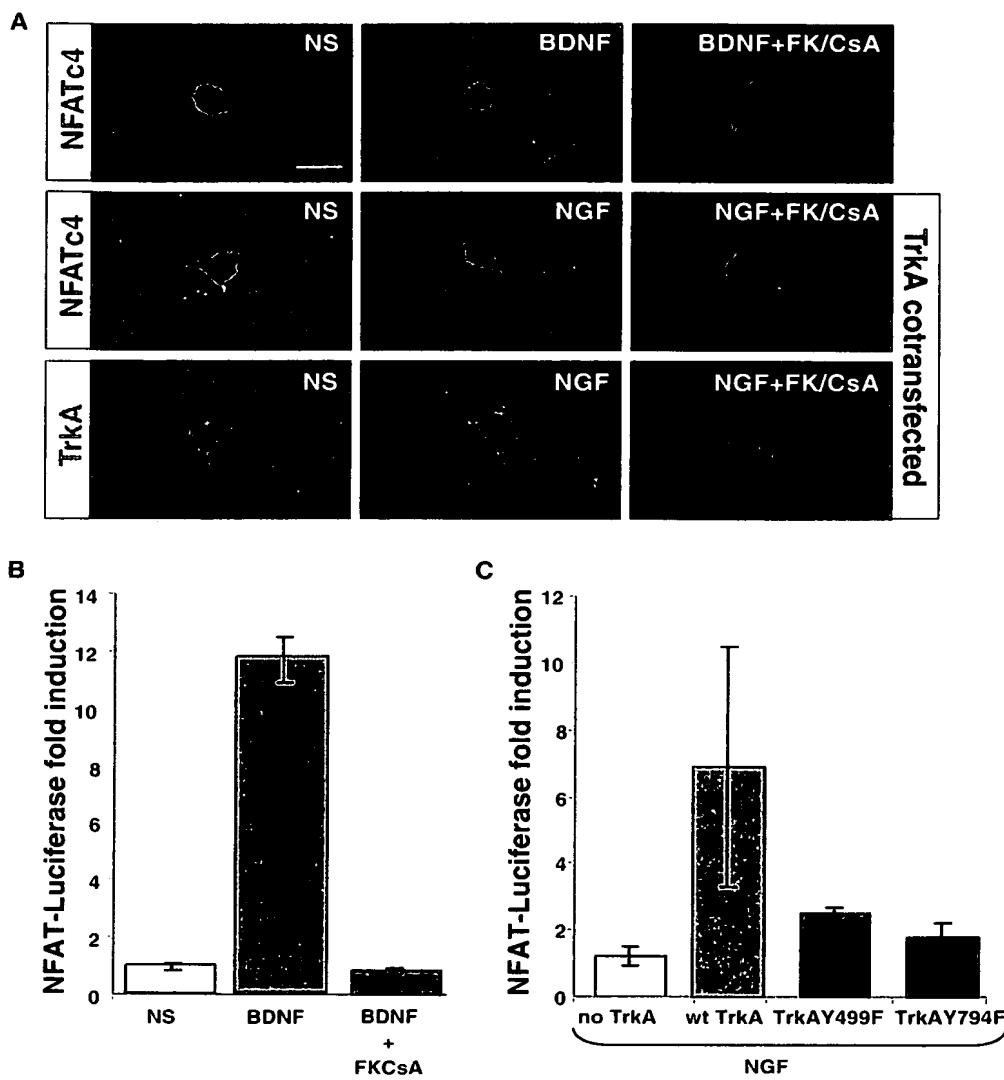
**Neurite Outgrowth Arrest after Calcineurin Inhibition  
Occurs with a Several Hour Delay**



**Figure 5 of 16**

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**Neurotrophins Regulate NFATc Translocation and  
Transcriptional Activation**



**Figure 6 of 16**

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**Inhibition of Calcineurin Blocks Netrin-dependent  
Growth of Dorsal Spinal Cord Explants**

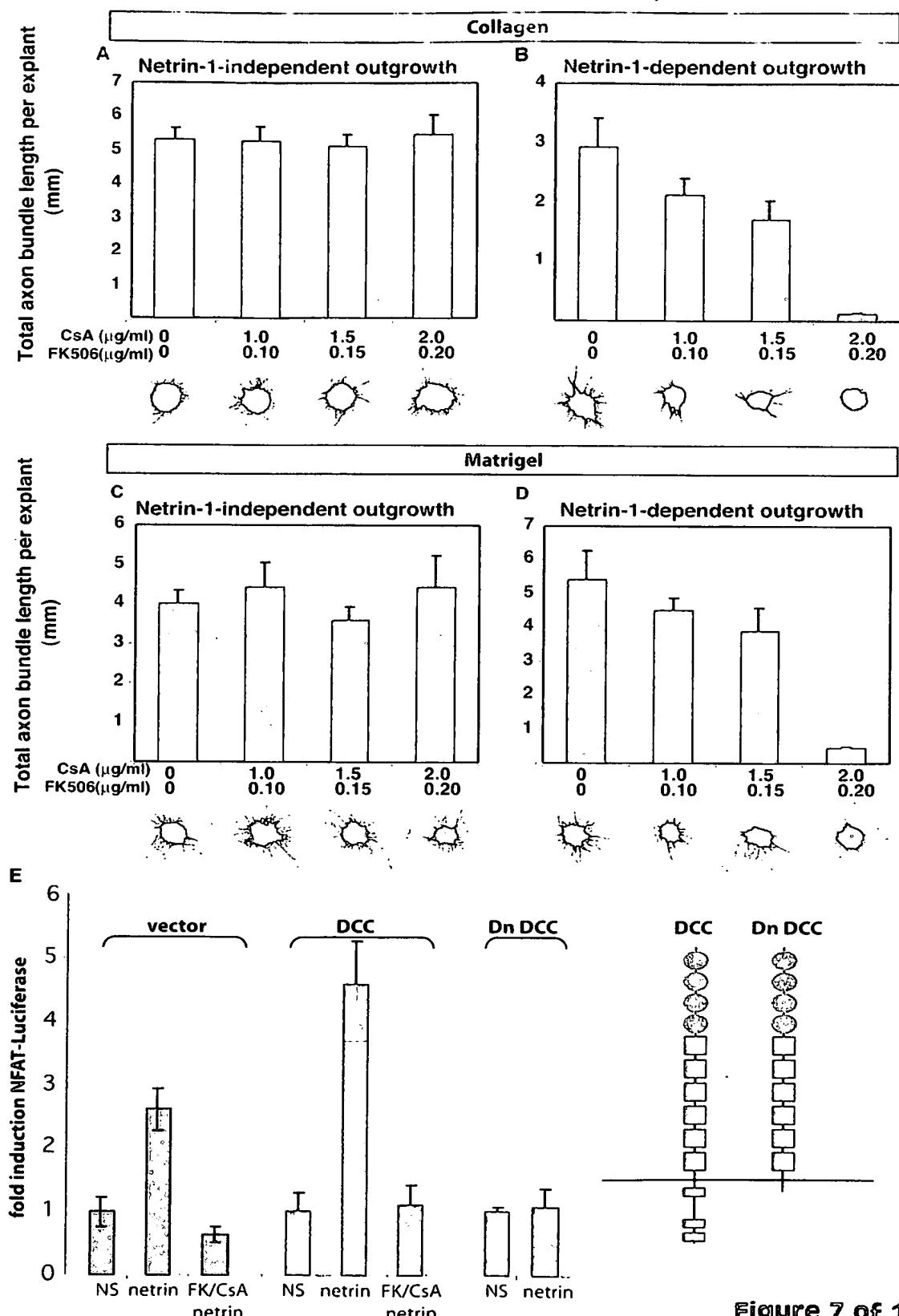


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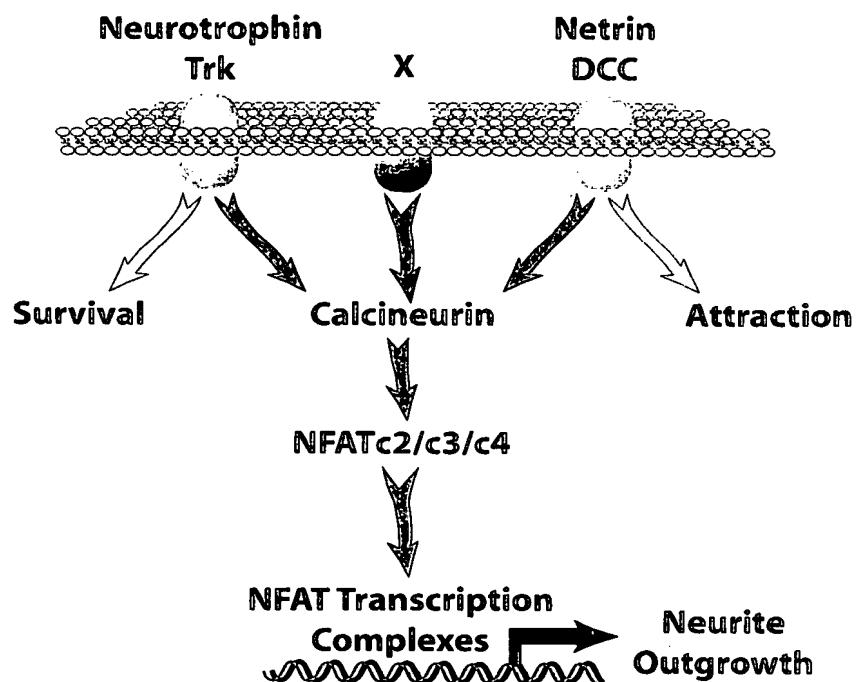
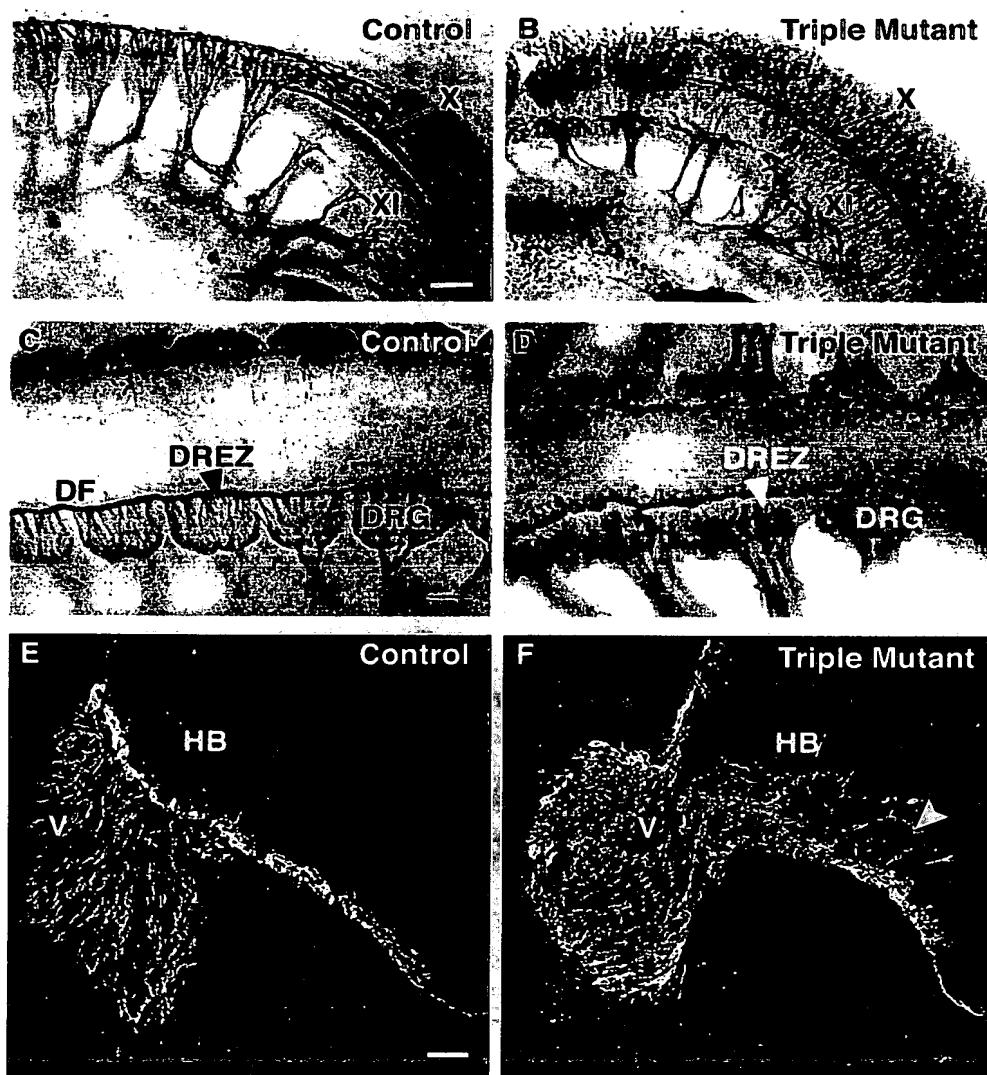


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**Axon Guidance Defects in NFATc2/3/4 Mutant Embryos**



**Figure 9 of 16**

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Normal Cell Type Differentiation of NFATc2/c3/c4 Mutant Neurons

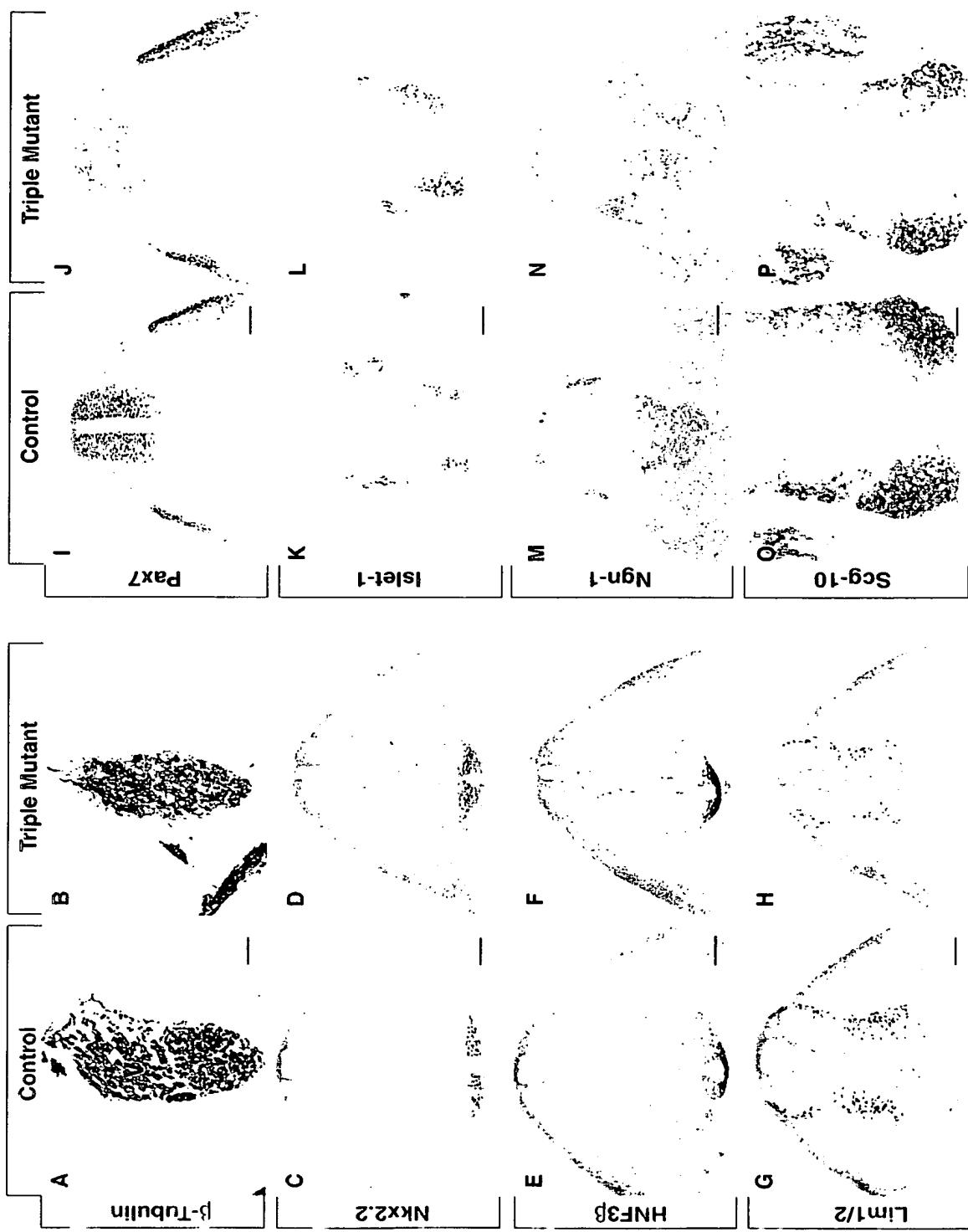
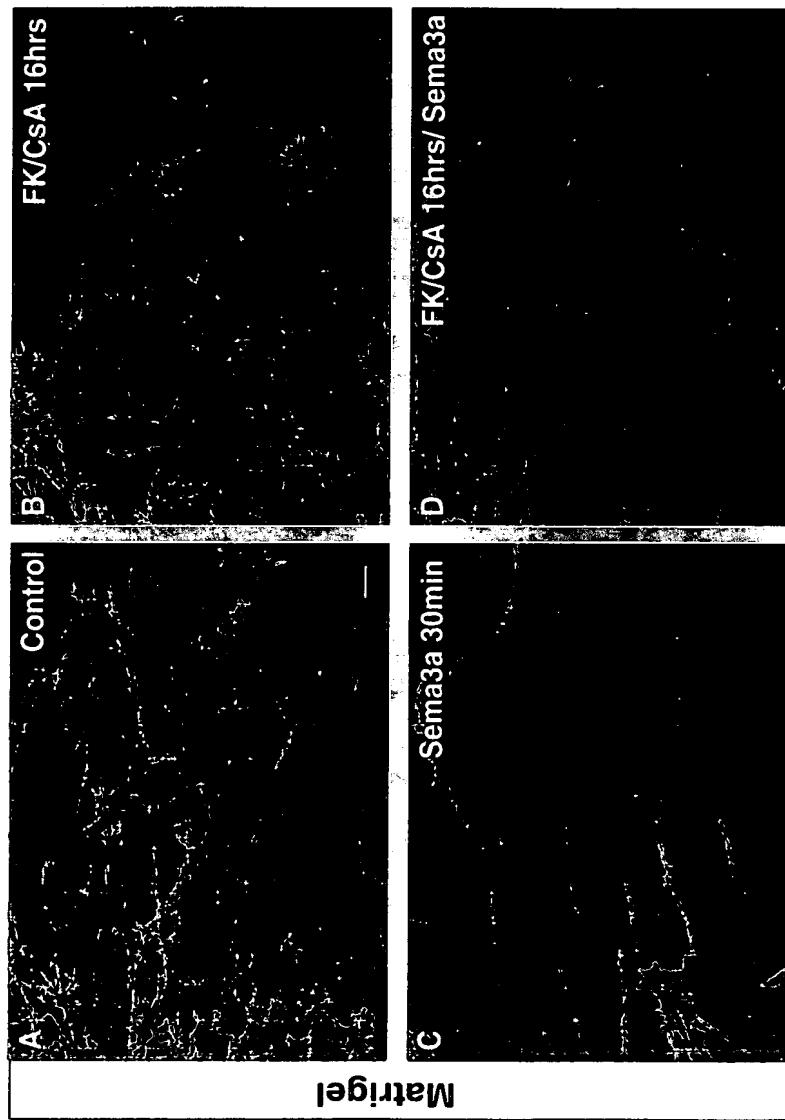


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**Inhibition of the NFATc/Calcineurin Pathway Does Not Affect  
Semaphorin-Induced Growth Cone Collapse**



**Figure 11 of 16**

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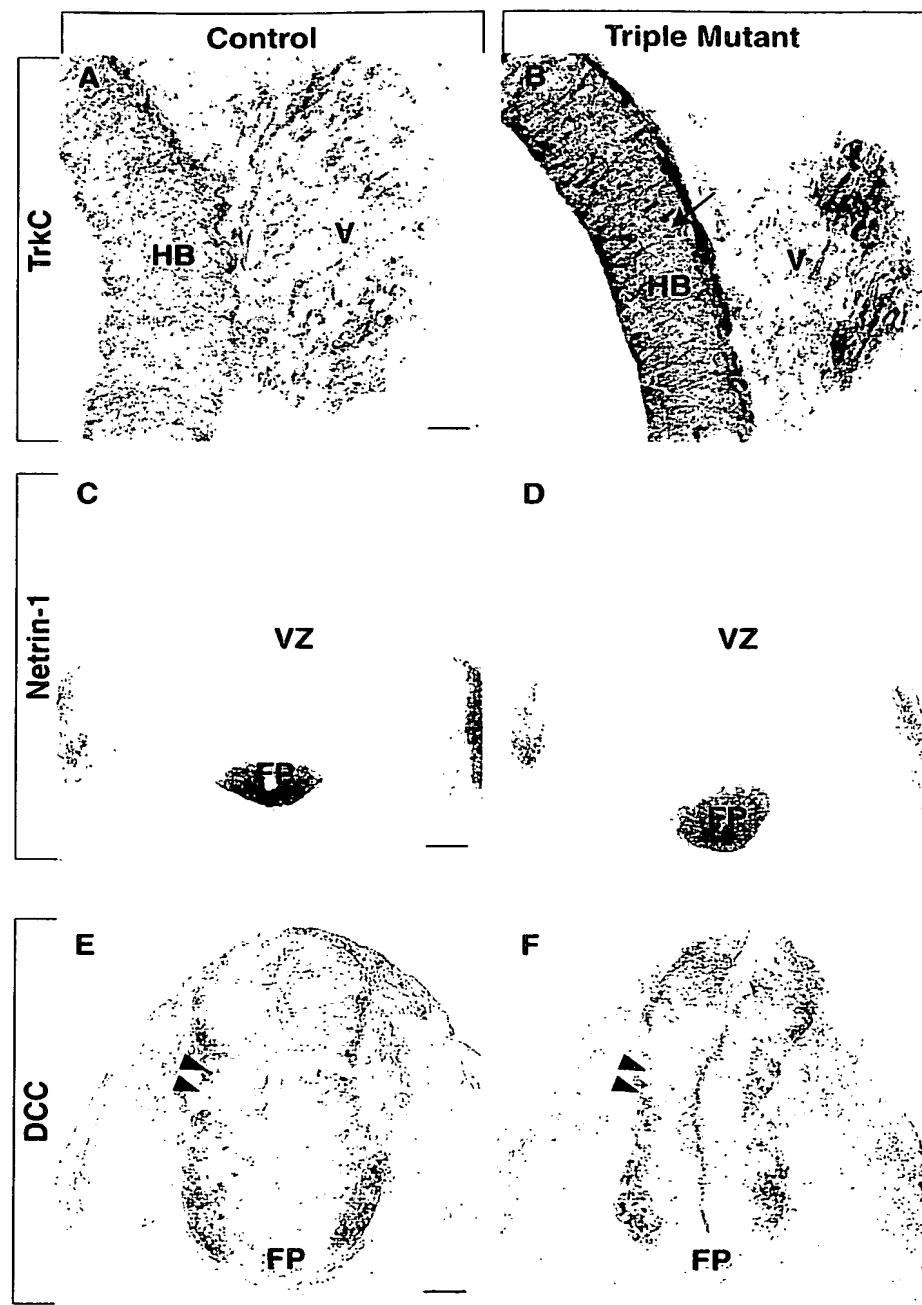


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